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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/899,059	07/06/2001	Guo-Liang Yu	075977-0122	5121

7590 09/05/2007

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EXAMINER
ROMEO, DAVID S

ART UNIT	PAPER NUMBER
1647	

MAIL DATE	DELIVERY MODE
09/05/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/899,059	YU ET AL.	
	Examiner	Art Unit	
	David S. Romeo	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *14 June 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-17,29 and 35* is/are pending in the application.

4a) Of the above claim(s) *8-10,29 and 35* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-7 and 11-17* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☒ Claim(s) *1-17,29 and 35* are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *06 July 2001* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claims 1–17, 29 and 35 are pending.

Applicant's election with traverse of group I, claims 1–17 and 29 in the reply filed on 06/14/2007 is acknowledged. The traversal is on the ground(s) that the search and examination of the two groups of claims is not unduly burdensome to the Examiner. This is not found persuasive because an application may properly be required to be restricted to one of two or more claimed invention if they are able to support separate patents and they are either independent (MPEP § 806.04 - § 806.04 (j)) or distinct (MPEP § 806.05 - § 806.05(i)). Groups I and II are distinct for the reasons given in the Office action mailed 02/14/2007. Furthermore, separate classification (i.e., class and subclass) of distinct inventions is sufficient to establish a prima facie case that the search and examination of the plural inventions imposes a serious burden upon the Examiner. See M.P.E.P. § 803. Such separate classification is set forth in the Office action mailed 02/14/2007.

The requirement is still deemed proper and is therefore made FINAL.

Applicant's election of the "inflammatory bowel disease" species in the reply filed on 06/14/2007 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

Claim 35 is withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 06/14/2007.

Claims 8–10 and 29 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected species, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 06/14/2007.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 3–7 and 13–16 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treatment comprising administering an anti-TNF-γ-β antibody, wherein the TNF-γ-β protein consists of amino acid residues 72-251 of SEQ ID NO: 20, does not reasonably provide enablement for a method of treatment comprising administering an anti-TNF-γ-β antibody, wherein the TNF-γ-β protein consists of the amino acid sequence of the polypeptide encoded by ATCC Deposit number 203055 or the amino acid sequence of the extracellular domain of the polypeptide encoded by ATCC Deposit number 203055. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

ATCC Deposit No. 203055 is required in order to make and/or use the claimed invention. The deposit rules (37 CFR 1.801 - 1.809) set forth conditions of deposit which must be satisfied in the event a deposit is required. 37 C.F.R. § 1.808(a)(2) requires that a deposit must be made under conditions that assure that, subject to paragraph (b) of this section, all restrictions imposed by the depositor on the availability to the public of the deposited material will be irrevocably

removed upon the granting of the patent. According to M.P.E.P. 2410.01, the mere indication that a deposit has been made under conditions prescribed by the Budapest Treaty would satisfy all conditions of these regulations except the requirement that all restrictions on access be removed on grant of the patent.

The specification acknowledges deposit of ATCC Deposit No. 203055 under conditions prescribed by the Budapest Treaty (paragraph [0132]). However, the mere indication that a deposit has been made under conditions prescribed by the Budapest Treaty would not satisfy the requirement that all restrictions on access be removed on grant of the patent. A provision that, with the one possible exception in 37 CFR 1.808(b), all restrictions on the accessibility of the deposit will be irrevocably removed by the applicant upon the granting of a patent is required.

Claims 1, 2, 11, 12 and 17 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claims are directed to or encompass an antibody or fragment thereof that binds "TNF-γ-β protein." The examiner uses the following passages from the specification to construe the term "TNF-γ-β protein":

> [0016] In accordance with all aspects of the invention, the term "TNF-gamma" refers to TNF-gamma-alpha and/or TNF-gamma-beta.
>
> [0173] The present invention is also directed to nucleic acid molecules comprising, or alternatively, consisting of, a polynucleotide sequence at least 80%, 85%, 90%, 92%, 94%, 95%, 96%, 97%, 98% or 99% identical to the

> polynucleotide sequences encoding the TNF-gamma-beta polypeptides described above, and the polypeptides encoded thereby.
>
> [0182] Thus, the TNF-gamma of the present invention may include one or more amino acid substitutions, deletions or additions, either from natural mutations or human manipulation.

Thus, the claims are directed to or encompass a genus of TNF-γ-β proteins.

An applicant complies with the written-description requirement by describing the invention, with all its claimed limitations, not that which makes it obvious, and by using such descriptive means as words, structures, figures, diagrams, formulas, etc., that set forth the claimed invention.

The specification provides a full-length TNF-γ-β polypeptide comprising the amino acid sequence of SEQ ID NO: 20 and several N-terminal deletions thereof ([0165]-[0166]). However, none of these constructs varies the amino acid sequence of SEQ ID NO: 2, other than by deletion, and thus these constructs are not representative of the genus. The specification and claim do not indicate what distinguishing attributes are shared by the members of the genus. The specification and claim do not place any limit on the number of amino acid substitutions, deletions, insertions and/or additions that may be made to SEQ ID NO: 20. Thus, the scope of the claim includes numerous structural variants, and the genus is highly variant because a significant number of structural differences between genus members is permitted. No common structural attributes identify the members of the genus. Since the disclosure fails to describe the common attributes or characteristics that identify members of the genus, and because the genus is highly variant, SEQ ID NO: 20 and several N-terminal deletions thereof alone are insufficient to describe the genus. One of skill in the art would reasonably conclude that the disclosure fails

to provide a representative number of species to describe the genus. Thus, applicant was not in possession of the claimed method.

Claims 1–7 and 11–17 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treatment, does not reasonably provide enablement for a method of prevention. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

The claims are directed to or encompass a method of preventing an inflammatory response, disease or disorder. The specification provides that:

> [Example 36] TNF-gamma-beta treatment of PBMCs results in proliferation of T cells and a significant increase in IFN-gamma production compared to controls.
>
> [Example 37] TNF-gamma-beta Exacerbates an In-vivo MLR Reaction.
>
> [0952] Thus, antagonists of TNF-gamma-beta (e.g., neutralizing antibody against TNF-gamma or soluble DR3 or TR6 proteins such as Fc or albumin fusion proteins) might have therapeutic potential in treating patients with this and other T cell-mediated inflammatory processes and diseases, including, but not limited to, systemic lupus erythematosus, multiple sclerosis, arthritis, and delayed-type hypersensitivity reactions.

However, there are no working examples of prevention. The examiner is aware that working examples are not required. Lack of a working example, however, is a factor to be considered, especially in a case involving an unpredictable and undeveloped art.

According to applicants' specification:

> [0492] In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to direct an individual's immune system towards development of a humoral response (i.e. TH2) as opposed to a TH1 cellular response.

> [0734] It is clear that most immune cells and cancer cells can express more than one TNF receptor (even more than one death receptor) and ligand superfamily member. The existence of multiple receptors for one ligand or multiple ligands for one receptor, and multiple splicing variant forms of receptor or ligand suggests an unexpected complexity in the regulation of apoptosis and immune function. These receptors and ligands appear to be functionally redundant, but their expression patterns are different, suggesting a distinct tissue or cell specific involvement in a particular function. Moreover, the expression of these ligands and receptors may differ at the level of individual cell types within tissues and the expression level on the same cell type may also differ.

> [0736] As shown in the table, DR3 and two forms of TNF-gamma are differentially expressed in different tissues and cells. In the libraries tested, DR3 was found to be expressed in most tissues, in activated T-cells, monocytes, dendritic cells, TH2 cells, and several other cell lines (such as U937, HeLa) and tumor tissues (such as hepatocellular tumor and Hodgkin's lymphoma).

According to Zhang (J Clin Invest. 2001 Jun;107(11):1459-68), TR6-Fc (a TNF-γ-β inhibitor) only inhibits, but does not prevent, in vivo and ex vivo splenic alloactivation in mice (Figure 3).

> We noticed that inhibition of proliferation in the mouse in vivo MLR (the graft-versus-host response) by TR6 was not complete even with high concentrations of TR6. The same was also true for the effect of TR6 on IL-2 production in in vitro MLR and on graft rejection in vivo. Further increase of the TR6-Fc concentration did not augment the degree of inhibition. This is probably due to the fact that costimulation via TR2 is only a part of the costimulation program for the T cells. Considering that CD28 plays a dominant role in costimulation, and that multiple costimulation pathways coexist as discussed earlier here, the partial inhibition is within our expectation.

Page 1466, right column, full paragraph 1.

According to Feldman (Transplant Proc. 1998 Dec;30(8):4126-7):

> … in a chronic immune-driven inflammatory response there are a number of pathways that become engaged and can serve to sustain the inflammatory process.

Page 4126. right column, last full paragraph.

In view of the breadth of the claims and the unexpected complexity in the art, and the facts that there are no working examples of prevention, that inhibition with a TNF-γ-β inhibitor was not complete even with high concentrations of the inhibitor, that multiple T-cell costimulation pathways coexist, that in a chronic immune-driven inflammatory response there are a number of pathways that become engaged and can serve to sustain the inflammatory process, the examiner concludes that it would require undue experimentation for the skilled artisan to make and/or use the full scope of the claimed invention.

Claims 1–7 and 11–17 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treating an inflammatory response, disease or disorder comprising administering a neutralizing or antagonistic antibody that specifically binds a TNF-γ-β protein, does not reasonably provide enablement for a method of treating an inflammatory response, disease or disorder comprising administering an antibody that specifically binds a TNF-γ-β protein. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

The claims are directed to or encompass a method of treatment utilizing any and/or all antibodies that specifically bind TNF-γ-β. The claims do not require a neutralizing or antagonistic antibody. As one of skill in the art would appreciate, a neutralizing or antagonistic anti-TNF-γ-β antibody would be required in order to achieve the desired treatment. See, for example, Kelly (J Clin Invest. 1996 Feb 15;97(4):1056-63), paragraph bridging pages 1056-1057:

> Antibodies vary in their specificity. Blocking a particular epitope does not necessarily neutralize the functional activity of the protein to which the antibody is directed.

The specification lacks guidance for, and working examples of, treating or preventing any and/or all inflammatory responses, diseases, or disorders with an antibody that does not block the functional activity of the protein to which the antibody is directed. The examiner is aware that working examples are not required. Lack of a working example is, however, a factor to be considered.

In view of the breadth of the claims, the limited amount of direction and working examples provided by the inventor, and the fact that blocking a particular epitope does not necessarily neutralize the functional activity of the protein to which the antibody is directed it would require undue experimentation for the skilled artisan to make and/or use the full scope of the claimed invention.

Claims 1–7 and 11–17 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treating inflammatory bowel disease, does not reasonably provide enablement for a method of treating an inflammatory response, disease or disorder or for a method treating an autoimmune disease or disorder. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

The claims are directed to or encompass the treatment of any and/or all inflammatory or autoimmune responses, diseases or disorders.

According to applicants' specification:

> [0492] In another specific embodiment, polypeptides, antibodies, polynucleotides and/or agonists or antagonists of the present invention are used as an agent to direct an individual's immune system towards development of a humoral response (i.e. TH2) as opposed to a TH1 cellular response.

According to Bamias (Proc Natl Acad Sci U S A. 2006 May 30;103(22):8441-6),

> The differentiation of naïve CD4+ lymphocytes into IFN-γ-secreting Th1 "effector" cells is a multistep process that involves several cell types, costimulatory molecules, transcription factors, and secreted cytokines … . Antigen-presenting cell (APC)-derived IL-12 is essential for the induction of IFN-γ, an effect that is greatly enhanced by IL-18 … . IL-12 up-regulates T-bet, a transcription factor that is critical for the stabilization of a T helper (Th)1-polarized phenotype … . Recently, additional cytokines that play prominent roles during Th1 responses have been described, such as IL-27 and IL-23 … . Engagement of the T cell receptor (TCR) provides further signals for the induction of IFN-γ, both in parallel to and independently of cytokine-mediated pathways … .
>
> Members of the TNF and TNF-receptor superfamilies of proteins (TNFSFPs and TNFRSFPs, respectively) are abundantly expressed in the immune system, and are critically involved in the differentiation, proliferation, and apoptosis of immune cells … . Several members of these families induce secretion of IFN-γ upon ligand/receptor binding, thereby enhancing Th1-type responses … . TL1A (TNFSPF15) is a recently identified, TNF-like factor that is currently the only known ligand for death-domain receptor (DR)3 … , which is primarily expressed on activated lymphocytes … .
>
> Binding of TL1A to DR3 triggers proliferative/activation signals, most likely through activation of NF-κB-mediated pathways … . TL1A specifically induces secretion of IFN-γ- by human T cells … , raising the possibility that TL1A/DR3 may participate in Th1-mediated responses. Indeed, we and others have recently reported up-regulation of both TL1A and DR3 in inflammatory bowel disease (IBD), particularly Crohn's disease (CD) … . Whereas original reports indicated that TL1A expression was confined to endothelial cells … subsequent studies demonstrated that in involved intestinal tissue from patients with IBD, TL1A was also expressed on lymphocytes, plasma cells, and monocytes. TL1A may participate in the pathogenesis of IBD, likely by inducing secretion of IFN-γ from lamina propria mononuclear cells (LPMCs) and the subsequent generation of proinflammatory responses … .
>
> Finally, we report that TL1A induces the proliferation of memory but not naïve CD4+ lymphocytes, a finding that supports a role for TL1A during the effector phase of Th1 responses.

Page 8441, left column, paragraph 1 through right column, full paragraph 2.

> Finally, TL1A preferentially acts on memory cells, which indicates that TL1A may be particularly important during the late/effector phase of Th1 immunity, increasing the amount of IFN-γ that is available and, thereby, increasing the magnitude of the immune response.

Page 8445, left column, full paragraph 2.

> [Crohn's disease] is considered a prototypic Th1-mediated condition in which IFN-γ plays a central pathogenetic role.

Page 8445, paragraph bridging left and right columns.

> We therefore conclude that interactions between TL1A and DR3 participate in the pathogenesis of Th1-mediated inflammation, including the inflammation observed in patients with CD.

Page 8445, right column, last full paragraph.

According to Tang (J Immunol. 2001 Feb 1;166(3):1471-81):

> In the mucosal immune system, resident dendritic cells are specialized for priming Th2-polarized immunity, whereas the Ag-presenting activity of macrophages has been linked with the development of Th1 phenotype. As an immune switch toward Th1 can protect against Th2-mediated allergic response, this study investigated the capacity of lung macrophages to stimulate Th1 responses during the secondary exposure to inhaled allergen, thereby suppressing Th2-mediated allergic airway inflammation in a murine model of allergic asthma. Following airway macrophage depletion in OVA-sensitized mice, lung T cells defaulted to a phenotype that produced less Th1 (IFN-) and more Th2 (IL-4 and IL-5) cytokines, leading to more severe airway hyperreactivity and inflammation after intranasal Ag challenge. After OVA pulsing and adoptive transfer, lung macrophages selectively promoted a Th1 response in Ag-sensitized recipients and did not induce pulmonary eosinophilia. By contrast, OVA pulsing and adoptive transfer of a lung cell preparation, consisting of dendritic cells, B cells, and macrophages, promoted a Th2 response with an associated inflammatory response that was suppressed when macrophages were present and pretreated with IFN-, but exacerbated when macrophages were depleted before IFN- treatment. In addition, Th1-promoting activity of lung macrophages was not related to the autocrine production of IL-12p40. These results suggest that the Th1-promoting APC activity may be an inherent property of the lung macrophage population, and may

play an important role, upon stimulation by IFN-γ, in antagonizing an ongoing Th2 immunity and Th2-dependent allergic responses.

According to Charlton (Curr Opin Immunol. 1995 Dec;7(6):793-8) autoimmune responses, diseases, and disorders can result from either Th1 responses (section bridging pages 793-794) or Th2 responses (page 794, section bridging left and right columns).

The specification lacks guidance for, and working examples of, treating Th2-mediated inflammatory or autoimmune responses, diseases and disorders. The examiner is aware that working examples are not required. Lack of a working example is, however, a factor to be considered.

In view of the breadth of the claims, the limited amount of direction and working examples provided by the inventor, and the fact that interactions between TL1A and DR3 participate in the pathogenesis of Th1-mediated inflammation, it would require undue experimentation for the skilled artisan to make and/or use the full scope of the claimed invention.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1, 2, 11, 12 and 17 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1, 2, 11, 12 and 17 are indefinite because they recite the term "TNF-γ-β protein." Because the instant specification does not identify that material element or combination of elements which is unique to, and, therefore, definitive of a "TNF-γ-β protein" an artisan cannot

determine what additional or material limitations are placed upon a claim by the presence of this element. The metes and bounds are not clearly set forth.

Conclusion

No claims are allowable.

ANY INQUIRY CONCERNING THIS COMMUNICATION OR EARLIER COMMUNICATIONS FROM THE EXAMINER SHOULD BE DIRECTED TO DAVID S. ROMEO WHOSE TELEPHONE NUMBER IS (571) 272-0890. THE EXAMINER CAN NORMALLY BE REACHED ON MONDAY THROUGH FRIDAY FROM 9:00 A.M. TO 5:30 P.M. IF ATTEMPTS TO REACH THE EXAMINER BY TELEPHONE ARE UNSUCCESSFUL, THE EXAMINER'S SUPERVISOR, MANJUNATH RAO, CAN BE REACHED AT (571)272-0939.

IF SUBMITTING OFFICIAL CORRESPONDENCE BY FAX, APPLICANTS ARE ENCOURAGED TO SUBMIT OFFICIAL CORRESPONDENCE TO THE CENTRAL FAX NUMBER FOR OFFICIAL CORRESPONDENCE, WHICH IS (571) 273-8300.

CUSTOMERS ARE ALSO ADVISED TO USE CERTIFICATE OF FACSIMILE PROCEDURES WHEN SUBMITTING A REPLY TO A NON-FINAL OR FINAL OFFICE ACTION BY FACSIMILE (SEE 37 CFR 1.6 AND 1.8).

ANY INQUIRY OF A GENERAL NATURE OR RELATING TO THE STATUS OF THIS APPLICATION OR PROCEEDING MAY BE OBTAINED FROM THE PATENT APPLICATION INFORMATION RETRIEVAL (PAIR) SYSTEM. STATUS INFORMATION FOR PUBLISHED APPLICATIONS MAY BE OBTAINED FROM EITHER PRIVATE PAIR OR PUBLIC PAIR. STATUS INFORMATION FOR UNPUBLISHED APPLICATIONS IS AVAILABLE THROUGH PRIVATE PAIR ONLY. FOR MORE INFORMATION ABOUT THE PAIR SYSTEM, SEE HTTP://PAIR-DIRECT.USPTO.GOV. CONTACT THE ELECTRONIC BUSINESS CENTER (EBC) AT 866-217-9197 (TOLL-FREE) FOR QUESTIONS ON ACCESS TO THE PRIVATE PAIR SYSTEM,

/DAVID ROMEO/
PRIMARY EXAMINER
ART UNIT 1647

DSR
SEPTEMBER 3, 2007